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Gastric Volvulus Complicating Paraesophageal Hiatal Hernia

ANTHONY B. SOKOL, M.D.,
Beverly Hills, AND
LEON MORGENSTERN, M.D., *Los Angeles*

GASTRIC VOLVULUS COMPLICATING a paraesophageal hiatal hernia is rare.[1,2] Because the threat of strangulation and infarction of the incarcerated stomach is great, prompt recognition and surgical correction of this condition is mandatory. Two cases occurring within a 14-day period at the Cedars-Sinai Medical Center prompted this review.

Reports of Cases

Case 1. A 68-year-old white woman with a known paraesophageal hiatal hernia had sudden onset of severe midepigastric pain during dinner. It radiated to the umbilicus and the back, remained steady and severe and was unrelieved by change of position or by taking food. When first seen in the hospital, she was restless and was noted to be obese. Despite complaint of severe abdominal pain, abdominal findings were negative. Attempts at passing a nasogastric tube were unsuccessful. An x-ray film of the chest showed double fluid levels and de Lorimier's hair pin loop with incisura directed toward the right upper quadrant (Figures 1 and 2). Two hours after admission a mass became palpable in the epigastrium and the patient was taken to surgery. At operation gastric volvulus was observed and the stomach, which had herniated through a 10 x 15 cm diaphragmatic defect, was incarcerated in the left side of the chest. As the patient's cardiovascular status began to deteriorate during the operation, as manifested by low blood pressure and irregular pulse, simple reduction of the volvulus was carried out without repair of the hiatal hernia. The postoperative course was complicated by a left pleural effusion

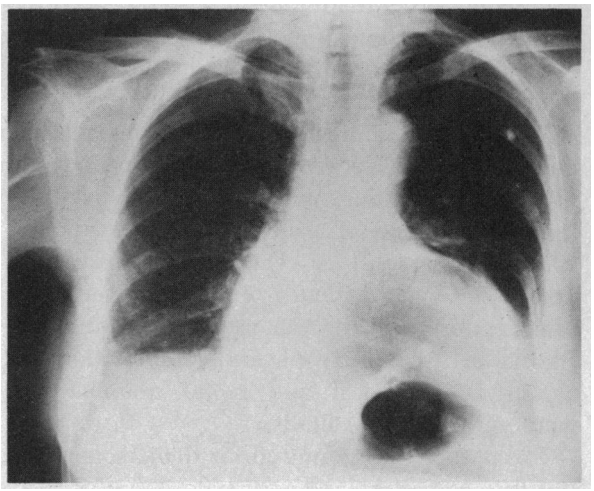

Figure 1.—(Case 1) Film of chest showing a double air level, one above and one below the diaphragm. This condition occurs when the fundic portion of the intrathoracic stomach redescends into the abdomen, leading to a closed loop phenomenon. Note the "hairpin loop" sign with the incisura directed toward the right upper quadrant.

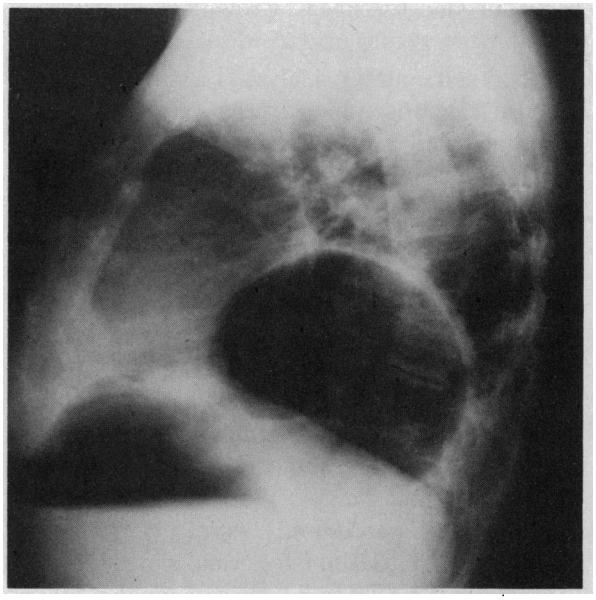

Figure 2.—Lateral view of the chest better demonstrating the double air fluid level shown in Figure 1.

From Cedars-Sinai Medical Center, Cedars of Lebanon Hospital Division, Los Angeles.

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Reprint requests to: L. Morgenstern, M.D., Department of Surgery, Cedars of Lebanon Hospital, 4833 Fountain Avenue, Los Angeles, Ca. 90029.

but was otherwise unremarkable. She became asymptomatic and elected not to undergo further operation.

Case 2. A 63-year-old white man entered the hospital with history of several days of persistent vomiting and abdominal pain with radiation to the back and lower left anterior chest, unrelieved by change of position or by antacids. There was a ten-year history of an "upside-down stomach." The patient appeared acutely ill, as characterized by anxiety, ashen facies and restlessness. He was severely dehydrated and complained of abdominal pain. There were no other significant physical findings. Laboratory work-up revealed severe hypochloremic, hypokalemic alkalosis, and hypovolemia. Upper gastrointestinal x-ray studies revealed gastric volvulus with a portion of the fundus incarcerated in the chest (Figure 3). After 48 hours, the fluid and electrolyte imbalance had been sufficiently corrected to permit operative intervention. At operation, because of labile blood pressure, the herniated cardiac and fundic portions of the stomach were simply reduced, as they appeared viable, and gastrostomy was performed. Eleven days later an operation for definitive transabdominal repair of hiatal hernia was carried out.

Symptoms of Acute Volvulus

Historically, the acute obstruction occasioned by volvulus usually begins not long after a meal, but it may awaken the patient from sleep. Usually there is history of several previous episodes that were relieved by non-specific measures.[3,4,5,6]

The patient complains of sudden epigastric or left lower thoracic pain with radiation through to the back, unrelieved by change of position. Associated with the pain are unsuccessful attempts at vomiting and symptoms of high gastrointestinal obstruction. On physical examination, although the patient is acutely ill, restless and complaining of severe pain, there are usually no actual physical findings, with the infrequent exception of possible tympany of the left lower thorax or a mass in the epigastrium. If the volvulus persists and does not spontaneously reduce, signs of cardiovascular collapse occur secondary to gastric infarction and progressive hypovolemia associated with diminished intake and the interstitial sequestration of fluid within the walls of the infarcted thoracic stomach.[4,7]

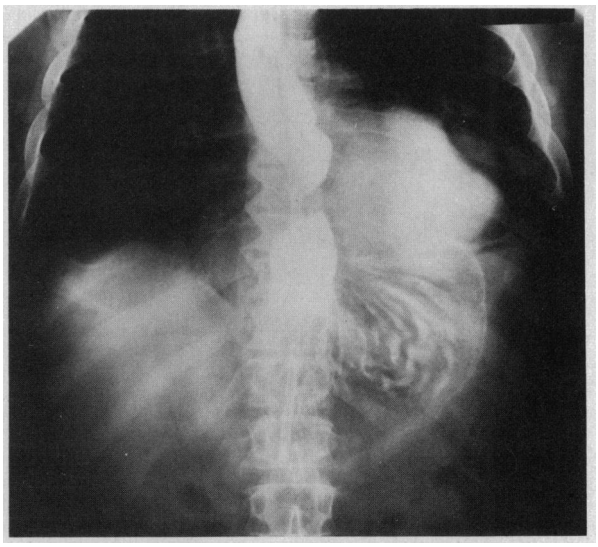

Figure 3.—(Case 2) Selected view from an upper gastrointestinal series demonstrating several signs of gastric volvulus. Note the rotation seen best by the rugal folds of the body radiating toward the lesser curvature. One can see the virtually complete pyloric obstruction secondary to the rotation, and edema is manifested by the lack of contrast material in the duodenum. Finally, note the vague shadow of contrast material in the chest which represented the incarcerated fundic portion of the stomach.

Diagnosis

Radiologic examination is diagnostic. Films of the chest or abdomen may demonstrate a high "simulated" left diaphragm which is actually the herniated and distended greater curvature of the stomach. Double air fluid levels occur if (as in Culver's cases and our own Case 1) the fundic portion of the thoracic stomach redescends into the abdomen.[4] There may be a "hairpin" loop with the incisura directed toward the right upper quadrant or posteriorly. Massive gaseous distension in the upper abdomen or chest may appear, with a gas bubble on either side of the midline. Barium swallow studies demonstrate the classic signs of volvulus, such as sharp cut-off of the esophageal or gastric barium column, abnormal twisting of rugal folds, and finally delineation of the intrathoracic portion of the stomach.

Feldman and de Lorimier's articles give excellent reviews as to the roentgenographic signs of gastric volvulus.[8,9,10]

Attempts at gastric intubation are either met with resistance due to obstruction at the esophagogastric junction or with initial relief of abdominal pain due to expulsion of great quantities of gas, only to be followed by progressive and per-

TABLE 1.—*Data on Eight Patients with Paraesophageal Hiatal Hernia* (PEHH)

Patient	*Age*	*Sex*	*Admission Date(s)*	*Symptoms of* PEHH	*Dx*	*Therapy and Findings*
1.	76	M	1965	Intermittent obstruct. assoc. with chest pain relieved with emesis	X-ray	Antacids for PEHH
2.	61	F	May 1963	Constricting chest pain	X-ray	Antacids for PEHH with partial incarceration of gastric fundus
			Aug 1963	Progressive abd pain, emesis for 3 days	Oper.	Operative reduction of a gastric volvulus with resection of an area of necrotic gastric fundus and repair of hiatal hernia
3.	73	F	1965	Epigastric gas and burning assoc. with meals	X-ray	Antacids for PEHH with esophagitis
4.	37	F	1962	Persistent chest pain and emesis	X-ray	Operative reduction of incarcerated PEHH containing stomach
5.	71	M	1962	Abdominal pain and gas	X-ray	Antacids for PEHH
			1963	None	Oper.	Resection of transverse colon
6.	36	F	Nov 1963	Regurgitation with bending over	X-ray	Operative repair of incarcerated PEHH
			Oct 1964	Recurrent symptoms	X-ray	Operative repair of recurrent incarcerated PEHH
7.	62	M	1963	Epigastric pain and nausea	X-ray	Antacids for PEHH
			1963	Persistent symptoms	Oper.	Operative reduction of incarcerated PEHH consisting of 50 percent of the stomach and the spleen. Because of high gastric acids, a P & V was performed in addition to repair of the hiatal hernia
8.	63	F	Feb 1963	None	X-ray	Antacids for bleeding duodenal ulcer
			Nov 1963	None	X-ray	Antacids for bleeding duodenal ulcer

sistent signs of the volvulus. Borchardt's and Lenormont's triad – unsuccessful vomiting, circumscribed epigastric pain, and inability to pass a tube into the stomach in a patient with a known paraesophageal hiatal hernia should make one suspect this dire complication.[1,4]

Treatment

Upon diagnosing gastric volvulus, operation should be performed immediately or after the least possible delay for correcting the associated fluid and electrolyte imbalance. Following reduction of the volvulus, definitive repair of the hiatal hernia will depend upon the clinical status of the patient.

The mortality of gastric volvulus is 40 percent, usually due first to lack of recognition and consequent profound cardiovascular collapse and second to the pneumonitic process which occurs from the intrathoracic infarcted stomach and repeated aspirations of esophageal secretions or foodstuffs.[1,2,5,10]

Incidence in Persons With Hiatal Hernia

During a five-year period, 77,231 patients were admitted to the private service of the Cedars of Lebanon Hospital. Of the 274 who were admitted for hiatal hernia, 206 had the sliding variety, 13 paraesophageal, and one the congenital type; fifty-four admissions were for "undiagnosed types" of hiatal hernia. In this category are included cases that could not be classified in the above categories after review of charts, radiographic reports and operative findings.

Discussion

The patients with the sliding type were found most frequently to have symptoms relating to reflux esophagitis and bleeding. Those with

paraesophageal hiatal hernia usually had obstructive symptoms (Table 1). While symptoms were mild in about half the cases of sliding type, they were severe in more than 75 percent of the paraesophageal type and 50 percent of the patients required operation.

Operative repair of paraesophageal hiatal hernia is recommended at its earliest stage whether symptoms exist or not.

Summary

Two cases of paraesophageal hiatal hernia were complicated by gastric volvulus. A distinct symptom complex occurs with obstruction and is associated with diagnostic radiographic criteria.

Of two hundred and seventy-four admissions to Cedars of Lebanon Hospital for hiatal hernia during a five-year period, thirteen were for paraesophageal hiatal hernia. Nine of the patients had symptoms and four required surgical repair.

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Primary Amebic Meningoencephalitis In California

ROBERT H. HECHT, M.D., AND ARTHUR H. COHEN, M.D., *Torrance*, AND JOHN STONER, M.D., *Los Angeles*, AND CHARLES IRWIN, M.D., *Torrance*

PRIMARY AMEBIC MENINGOENCEPHALITIS, to be distinguished from infections caused by Entamoeba histolytica, is a newly recognized disease entity. The infrequently encountered causative organism is usually a ubiquitous, free living, soil ameba of the Naegleriidae family (species Naegleria). The first cases were reported in 1965 from Australia[1] and since then, to our knowledge, 54 cases have been documented. In the United States, the disease has been detected in Florida,[2,3] Texas,[4] Virginia[5,6,7,8] and Georgia.[9] Elsewhere, this diagnosis has been made in Australia,[1,10] Czechoslovakia,[11,12] Britain,[13] Ireland,[13] New Zealand[14] and Africa.[15] This account will document the first reported occurrence of primary amebic meningoencephalitis in California and will describe its clinical, epidemiological and pathological characteristics. Furthermore, the entity will be reviewed to acquaint others with this rapidly fatal disease in order that it will be promptly recognized and early vigorous attempts at appropriate therapy undertaken. In addition, as new cases are recognized, measures should be instituted to prevent further outbreaks.

Report of a Case

A 16-year-old caucasian girl was admitted to Harbor General Hospital on April 29, 1971, with

From the Department of Pathology (Dr. Cohen), the Division of Infectious Diseases (Dr. Hecht), the Department of Medicine (Dr. Irwin), Harbor General Hospital, Torrance; and the Department of Medicine (Dr. Stoner), University of California, Los Angeles.

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Reprint requests to: A. H. Cohen, M.D., Harbor General Hospital, Department of Pathology, 1000 West Carson Street, Torrance, Ca. 90509.